ACUTE TOXICITY SUMMARY

MERCURY (INORGANIC)

Molecular Formula	Molecular Weight	Synonyms	CAS Registry Number
Hg°	200.59	mercury quicksilver colloidal mercury	7439-97-6
$HgCl_2$	271.52	mercuric chloride corrosive sublimate mercuric bichloride mercury perchloride	7487-94-7
$Hg_2(NO_3)_2$	525.19	mercurous nitrate mercury protonitrate	10415-75-5
$Hg(NO_3)_2$	324.66	mercuric nitrate mercury pernitrate	10045-94-0
Hg_2O	417.18	mercurous oxide mercury oxide	15829-53-5
HgO	216.59	mercuric oxide CI 77760 santar	21908-53-2
$HgSO_4$	296.68	mercuric sulfate mercury bisulfate	7783-35-9

I. Acute Toxicity Summary (1-hour exposure)

Inhalation reference exposure level	**1.8 µg/m³**
Critical effect(s)	behavioral deficits after in utero exposure to metallic mercury vapor
Hazard Index target(s)	Reproductive/developmental

II. Physical and Chemical Properties (HSDB, 1994)

Description	Hg: silver-white liquid Hg salts: white crystalline solids mercurous oxide: black powder
Density	$HgCl_2$: 5.44 g/cm^3 @ 25°C HgO: 9.8 g/cm^3 @ 25°C $HgSO_4$: 6.47 g/cm^3
Boiling point	Hg°: 356.7°C $HgCl_2$: 302°C
Melting point	Hg°: -38.9°C $HgCl_2$: 276°C

	$HgHNO_3$: 70°C
	$Hg(HNO_3)_2$: 79°C
Vapor pressure	Hg: 0.002 mm Hg @ 25°C
	$HgCl_2$: 1 mm Hg @ 136°C
Flashpoint	not applicable
Explosive limits	not applicable
Solubility	Hg°: 0.28 μmoles/L water @ 25 C
	$HgCl_2$: 69 g/L water, soluble in acetic acid.
	HgO: insoluble in water, soluble in nitric acid.
	$HgSO_4$: soluble in hydrochloric acid
	Most Hg_2^{2+} forms are water soluble.
	Hg^{2+} forms are not water soluble.
Odor threshold	odorless
Odor description	odorless
Metabolites	methylmercury (CH_3Hg)
Conversion factor	1 ppm = 8.34 mg/m³ (Hg° vapor) @ 25°C

III. Major Uses or Sources

Mercury compounds have a very wide range of chemical uses. Mercuric chloride is used as a wood preservative, a photographic intensifier, a dry battery depolarizer, a tanning agent for leather, and for separating lead from gold (HSDB, 1994). Mercuric nitrate is used in the manufacture of felt, and in the manufacture of bronze (HSDB, 1994). In addition to the above chemical uses, mercury is also released during municipal hazardous waste generation. Elemental mercury (Hg°) vapor is the dominant form in the atmosphere, followed by mercuric ionic species (Hg^{2+}) and methylmercury (CH_3Hg).

IV. Acute Toxicity to Humans

The respiratory tract is the first organ system affected in the case of acute inhalation poisonings (Levin *et al.*, 1988). Acute exposure to Hg° can lead to shortness of breath within 24 hours and a rapidly deteriorating course leading to death due to respiratory failure (Kanluen and Gottlieb, 1991).

In a case report, Kanluen and Gottlieb (1991) observed 4 individuals from a private home where silver was being smelted from dental amalgam containing an unknown amount of Hg°. All individuals died 9-23 days post-exposure from respiratory distress despite treatment with dimercaprol, a mercury chelator. At autopsy, necrotizing bronchiolitis with edema, emphysema, and insipissated fibrin was observed. Concentrations of mercury to which the individuals were exposed and duration of exposure were not estimated.

Central nervous system (CNS) effects such as tremors or increased excitability are sometimes seen in cases of acute accidental exposures (Goyer, 1993). Long-term effects from a single exposure to Hg° have been reported in 6 male workers exposed to an estimated concentration of 44 mg Hg/m³ for a period of several hours (McFarland and Reigel, 1978). Long-term CNS effects included nervousness, irritability, lack of ambition, and loss of sexual drive for several years. Shortness of breath also persisted for years in all cases. Similar cases of CNS disturbances, including irritability, insomnia, malaise, anorexia, fatigue, ataxia, and headache have been reported in children exposed to vapor from spilled elemental mercury in their home (Florentine and Sanfilippo, 1991).

Acute inhalation exposure of Hg° vapors from broken thermometers resulted in generalized skin eruptions in 15 individuals (Nakayama *et al*., 1983). The doses and durations of exposure were not estimated.

An infant exposed in an incubator to Hg° from a broken thermometer exhibited very high urinary (0.34 mg/L) and fecal (quantity not specified) mercury levels, but mercury levels in the blood were undetectable (McLaughlin *et al*., 1980). The infant recovered from the exposure within days with no lasting effects.

Mercury vapor is efficiently absorbed (76-100%) through inhalation via the nose over concentrations ranging from 4-30 $\mu g/m^3$ (Oikawa *et al*., 1982). Absorption is markedly decreased to 20% if the breathing is done only through the mouth. The biological half-life of mercury in the human brain is reported to be 21 days, and the half-life in the kidney is 64 days (Hursh *et al*., 1976). Mercury has been shown to accumulate in the placentae of women even in the absence of a particular history of mercury exposure (Suzuki *et al*., 1971).

Predisposing Conditions for Mercury Toxicity

Medical: Persons with preexisting allergies, skin conditions, chronic respiratory disease, nervous system disorders, or kidney diseases might have increased toxicity (Reprotext, 1999).

Chemical: Persons exposed to other neurotoxicants might have increased sensitivity (Reprotext, 1999).

Other: People who consume significant amounts of fish from areas with advisories for daily fish intake due to mercury contamination may be more susceptible to the acute toxicity of airborne mercury.

V. Acute Toxicity to Laboratory Animals

An LD_{50} value of 2.6 mg Hg/kg as mercuric nitrate by intravenous injection in chickens has been reported (HSDB, 1994).

Severe cellular degeneration and necrosis was observed in the kidneys, brain, colon, and heart tissue of 2 rabbits exposed for 4 hours to 29.7 mg Hg/m^3 (Ashe *et al*., 1953). Exposure of rabbits to 31.3 mg Hg/m^3 for 1 hour resulted in moderate pathological changes (unspecified), but no necrosis, in the brain and kidney; in contrast, heart and lung tissues showed mild pathologic changes (Ashe *et al*., 1953). Increased duration (6 hours/day for 5 days) of exposure at this concentration was lethal.

Livardjani *et al*. (1991) exposed 64 rats to 26 mg Hg/m³ for 1 hour, with an elevation in lung superoxide dismutase activity observed 3 days following exposure. Lethality was observed in 50% of rats 5 days following a 2-hour exposure to the same concentration.

Berlin and Johansson (1964) showed that inhaled mercury vapor (10 mg/m³ for 4 hours) resulted in a ten-fold greater brain burden of mercury in mice than the same dose administered intravenously (IV). Berlin *et al*. (1969) later showed that the inhalation route of exposure results in approximately 10-fold greater accumulation of mercury in the brain tissue of rats, rabbits, and monkeys than IV injection.

Induction of autoimmune glomerulonephritis, as measured first by elevated serum IgE levels and later by anti-laminin antibodies and proteinuria, has been observed in rats exposed to 1 mg Hg/m³, 6 hours/day, for 14 days (Hua *et al*., 1993).

VI. Reproductive or Developmental Toxicity

Mercury compounds, including inorganic forms, are listed under California Proposition 65 (Cal/EPA, Safe Drinking Water and Toxic Enforcement Act of 1986) as developmental toxins.

In rats, elemental mercury readily crosses the placental barrier and accumulates in the placenta following inhalation (Clarkson *et al*., 1972). Gale (1974) reported decreased crown-rump length and increased incidence of edema in hamster fetuses following single subcutaneous administration of 4 mg/kg Hg as mercuric acetate on day 8 of gestation. Exposure to 2.5 mg/kg Hg resulted in no significant developmental defects in these hamsters. Gale (1981) later showed that the most common manifestations of mercury-induced embryotoxicity in hamsters were resorption, edema, and cardiac abnormalities.

Pregnant rats exposed by inhalation to 1.8 mg/m³ of metallic mercury for 1 hour or 3 hours/day during gestation (days 11 through 14 plus days 17 through 20) bore pups that displayed significant dose-dependent deficits in behavioral measurements 3-7 months after birth compared to unexposed controls (Danielsson *et al*., 1993). Behaviors measured included spontaneous motor activity, performance of a spatial learning task, and habituation to the automated test chamber. The pups also showed dose-dependent, increased mercury levels in their brains, livers, and kidneys 2-3 days after birth.

VII. Derivation of Acute Reference Exposure Level and Other Severity Levels (for a 1-hour exposure)

Mild Adverse Effect Level

The most sensitive endpoint for inorganic mercury toxicity is developmental toxicity, which is considered a serious effect. Therefore, there is no mild adverse effect level for inorganic mercury.

Reference Exposure Level (protective against severe adverse effects): 1.8 μg/m³

Study	Danielsson *et al*., 1993
Study population	groups of 12 pregnant rats
Exposure method	inhalation of metallic mercury vapors
Critical effects	CNS disturbances in offspring
LOAEL	1.8 mg/m³
NOAEL	not observed
Exposure duration	1 hour per day
Extrapolated 1 hour concentration	1.8 mg/m³
LOAEL uncertainty factor	10
Interspecies uncertainty factor	10
Intraspecies uncertainty factor	10
Cumulative uncertainty factor	1,000
Reference Exposure Level	0.0018 mg/m³ (1.8 μg/m³)

Maternal rats exposed by inhalation to 1.8 mg/m³ of metallic mercury vapor for 1 hour/day or 3 hours/day during gestation bore offspring that displayed significant dose-dependent deficits in behavior 3-7 months after birth compared to controls. Since mercury salts have no significant vapor pressure under normal atmospheric conditions, they would only be of concern as hazards if aerosolized in aqueous solution or burned. This REL is developed for metallic mercury vapor and would be an overestimate of the REL for mercury salts.

Level Protective Against Life-threatening Effects

No recommendation is made due to the limitations of the database.

NIOSH lists a revised IDLH of 10 mg/m³ based on acute inhalation toxicity data of mercury vapor in animals (Ashe *et al*., 1953). Severe damage in the kidneys, lungs, and colon of rabbits resulted from exposure for a single 4-hour period to 28.8 mg/m³. Mild damage to most of the organs occurred after 1 hour of exposure. It is not clear why this result would make 10 mg/m³ a life-threatening level for humans.

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